UNITED STATES DISTRICT COURT

SOUTHERN DISTRICT OF OHIO

WESTERN DIVISION

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UNITED STATES OF AMERICA,	:	CASE NO. 1:07cr60(3)
Plaintiff,	:	Cincinnati, Ohio
- v -	:	Monday, March 14, 2011 1:32 p.m.
PAUL H. VOLKMAN,	:	
		DAY 8 OF JURY TRIAL
Defendant.	:	AFTERNOON SESSION

- - -

TRANSCRIPT OF PROCEEDINGS
BEFORE THE HONORABLE SANDRA S. BECKWITH, SENIOR JUDGE,
AND JURY

- - -

APPEARANCES:

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Also Present: Agent Christopher Kresnak (DEA)

Law Clerk: Laurie J. Nicholson, Esq.

Courtroom Deputy: Mary C. Brown

Court Reporter: Jodie D. Perkins, RMR, CRR

Proceedings reported by stenotype.
Transcript produced by computer-aided transcription.

AFTERNOON SESSION, Monday, March 14, 2011

(The jury entered the courtroom at 1:32 p.m.)

MICHAEL POLICASTRO, M.D.

being previously duly sworn, was examined and testified further as follows:

THE COURT: Welcome back, everyone. I think we're ready to resume.

Mr. Wright.

DIRECT EXAMINATION (Cont'd)

BY MR. WRIGHT:

Q. Doctor Policastro, before we move on to Scottie Lin James, I want to just step back, given that we've reviewed the circumstances of eight different patients, and I wanted to get your view as a toxicologist about whether or not the prescriptions that we see so far make any medical sense to you.

A. That's a difficult statement. The pain medication utilization would be prescribed. The combination of said hypnotics in high frequencies that are shorter than the half-lives does not make toxicologic sense.

Q. Okay. And why not?

A. Because of the increase in risk for adverse outcome. The combination -- utilization of opiates and sedatives dramatically increases your risk of adverse effect. When you look at drug and poisoning overdoses, particularly opiate related deaths, very rarely are they in isolation. Very rarely

is that a single drug, by itself, is very rarely associated with death. The combination of agents that we see, particularly when treating patients that have opiate related deaths, tend to be a combination of opiates and sedatives.

Q. In your daily job you're a consultant of sorts for other physicians in the hospital that are prescribing medications; is that right?

A. I am available for consultation, correct.

Q. And if someone said I want to prescribe two sedatives and two opiates, what would you tell them?

A. I would state that there's a significant risk for negative adverse outcome. For example, when we do procedures on patients, even if they are in chronic pain or acute pain, when I see patients that are on opiates and sedatives and I have to do procedures on those patients, we have to put these patients on oxygen monitor/cardiac monitor due to the risk of sudden death.

And when I say that, I mean the significant decline in your respiratory status in an unmonitored situation. Oftentimes these patients are already on pain medication, so giving them a medication on top of that for a procedure, we would have to utilize the modalities that I mentioned.

Q. So you would have to put -- if you were going to observe -- put somebody on these kinds of medications, you would need to put them on oxygen or at least monitor them in some way?

A. Correct.
Q. And why would that be?
A. Because of the sudden unexpected decline in respiration and oxygenation.
Q. You wouldn't just let them sit in the waiting room and see what happened?
MS. CROSS: Objection.
THE COURT: Overruled.
Q. Would you do that?
A. No.
Q. Why not?
A. Because of sudden risk of decline. After we perform a procedure on a patient when utilizing those medications, we observe them for a long period of time in the emergency department with continuous monitoring, as mentioned.
Q. And would that monitoring include professional staff that would be observing the patient?
A. Yes.
Q. And some kind of monitoring device to detect their breathing?
A. Correct. Their oxygenation, which is a marker of breathing, as well as with observation of their breathing status.
Q. And would the doctor ever come in and make sure that things were going okay?

A. The doctor being me?
Q. Yes.
A. Yes.
Q. We talked a good deal about the opiates. And I want to just ask you a few questions about the sedatives, in particular Soma.
Is Soma a medication that you typically would prescribe in your experience?
A. No.
Q. Why not?
A. Because Soma's utilization has been found that it has a highly abusive potential. It has significant euphoria and it is well recognized that, in the past, Soma, which is metabolized meprobamate, meprobamate was primarily seen as a drug of abuse. My clinical experience with this is that patients that request Soma are often patients that have a greater likelihood of drug abuse.
Q. Is Soma a medication that you have ever prescribed for pain purposes?
A. No.
Q. And you also talked a little bit about short-acting and long-acting medication. Could you just explain the difference again for the jury?
A. Short-acting is exactly what it sounds like -- it is short acting. Long-acting is longer acting. With regard to the use

of oxycodone, again, the long-acting form has -- you can think of it as two ways: There's a short phase, which is rapidly absorbed, and it lasts a long, long time over a period of time.

Q. And one last question just generally. In your review of the medical records, did you come across any documents that related to blood levels?

A. Did I come across any documents that relate to blood levels?

Q. Yes.

A. Yes.

Q. Describe those documents from what you remember.

A. The documents in relation to each patient or --

Q. Just the ones that were sent to the Mayo Lab.

A. So there were utilization of labs, both urine and blood. The problem is interpretation of specific levels on spot urinalysis. At times there was a urine sent for quantitative value, meaning that when the person urinated, they test the urine, said these drugs are present. And that's fine, saying that the drugs are present.

There was a quantitative value noted, which makes basically no sense in isolation. What you are trying to determine from this is through a series of urinalysis, analysis of several urine specimens, would be: Could you determine a rate of excretion to imply toxicity?

Q. Well, wait a minute. Let's back up. I mean, so you're

saying that the actual urine test might be able to show a presence?

A. It absolutely shows presence. It says this drug is present.

Q. Why couldn't it tell you if someone was taking their medication as prescribed?

A. Because all you have is that the urine is present. That value, in and of itself, cannot be attributed to compliance. All you can say is that the drugs were taken. The dosing regimen cannot be accurately obtained from it.

More importantly, in isolation from a single urine test, you can't determine dose compliance on it. It is meant for a spectrum. But even that is dependent upon too many factors. The urine clearance is added clearance, meaning from the liver, kidney, et cetera.

The other thing that it does not take into account, and which is the most important, is the combination of these drug agents. So, for example, a blood test that was sent for a meprobamate level, which is the metabolite of Soma, one of the sedatives, that drug concentration in itself assumes that you're just taking that one drug. It doesn't determine toxicity based off of the interaction of all of the other drugs.

Q. As a toxicologist, would you ever try to use a blood level to determine whether or not someone was taking three pills a

day as opposed to five pills a day?
A. No.
Q. Is that -- because in order to determine it, you would need so many tests over time to be able to get an accurate view?
A. There's too many factors to determine true dose compliance. It would require several periods of observation to determine what this person's plasma concentration is based off of their unique genetic mechanisms.
The other point too, as I mentioned, when you're dealing with multiple drug interactions, a single concentration is not going to be reflective of toxicity.
Q. So what did you make of the blood level reports that you saw in the medical records that were part of your review?
A. There were a number.
Q. Does it correlate with anything?
A. I cannot determine toxicity based off of that. As I mentioned, the urinalysis values could not be retrograde calculated to determine plasma concentration, which is basically, from each secretion in the urine, you could not determine a plasma concentration. And in addition, like I said, the meprobamate blood levels could not be used in isolation because several drugs were used. So even if that value is normal from the laboratory range reported, you can't determine is there multiple drug interactions.
Q. Meaning if there's more than one drug in the system, then

the numbers are even harder to interrupt?
A. Yes.
Q. Let's turn to Scottie Lin James, who died on September 30th, 2005. When was her last visit to Doctor Volkman?
A. 9/30/05.
Q. Are you sure?
A. I apologize. 9/26/05.
Q. And how many days after that did she die?
A. 9/30/05.
Q. And her age?
A. Thirty.
Q. And time of death?
A. 7:24 a.m.
Q. What were your sources of information regarding that?
A. The progress notes, the diagnostic tests, prescriptions, autopsy report, toxicology report and death certificate.
Q. What warning signs did you see, just based on your review of the medical records?
A. So in the review of the medical records, she had openly deceived to obtain narcotics. She stated that she had a history of ovarian cancer when, in fact, she had cervical dysplasia, which is a precancerous, at most, lesion of the cervix. This was not ovarian cancer. It was noted by Doctor Volkman and his staff that the patient openly deceived them and

she was dismissed from their practice.
Q. As a toxicologist and as a physician, why would that kind of deception concern you in deciding what to prescribe someone?
A. Again, that would imply a potential history of substance abuse and, again, their risk for adverse outcome would be greater than an individual that does not have those risk factors.
Q. Have you ever had someone try to fake cancer in order to get pain medication?
A. No.
Q. I'm going to ask you about the two visits as we go. The first visit, let's walk through what Ms. James received on September 16th, 2005.
A. She received two narcotic pain medications, which are oxycodone, Percocet, and two sedatives, which are Xanax and Soma.
Q. And you had talked earlier in the context of Mr. Jordan about something called toxicological chaos, and I wondered actually what you meant by that.
A. It is what I mentioned before, is that when you have dosing frequencies more often than the extent of half-life, it is virtually impossible to accurately depict what a peek plasma concentration will be. And as a result of that, the safety index of the drug declines.
Q. Now, let's look at the prescriptions that Ms. James

receives ten days later, on September 26th, 2005. What does she receive?
A. Oxycodone and Percocet, which are opiate pain medicines.
Q. And let's add these up in that ten-day period. What did Ms. James receive?
A. She received oxycodone, Percocet, Xanax and Soma.
Q. And how many pills was she supposed to take, according to these two inscriptions each day --
A. Forty-two.
Q. Should any human being be taking 36 pills a day of oxycodone?
A. I can't answer that specifically.
Q. Is that a prescription that you would typically see in your practice?
A. The number of tablets is very large. But, again, as I mentioned, the analgesic ceiling is not there. What would be important is with regards to frequency of that medication dispense.
Q. Is someone supposed to be taking oxycodone that frequently -- we'll move on.
Let's look at the autopsy report for Miss James. What did you find?
A. That she was found to have fluid in her lungs again and other organs were filled with fluid.
Q. What's visceral congestion?

A. Organs.
Q. Fluid in the organs?
A. It is just reflective of the congestion, overall.
Q. Okay. And how again does the opiate or how again do the drugs cause that kind of --
A. They trigger a reaction in the brain and the lungs to fill with fluid.
Q. And what did you find in the toxicology report?
A. Several things were actually pertinent. She had the presence of oxycodone, which was noted she was prescribed oxycodone. She had a vitreous level, as well as a blood level.

As I mentioned, oxycodone appears to come into the eyes slower than out of the eye. There's some correlation between the eye and the blood, again, these numbers are there.

Does postmortem redistribution occur and can values be elevated? Yes. So does she have presence of the drug? Yes.
Q. And then besides oxycodone -- just to be clear, both of those were within the range of levels associated with death?
A. Reported in the medical literature, yes. But they may be artificially elevated secondary to PMR, which is postmortem redistribution.
Q. What other drugs were present in Ms. James' system?
A. So she had a metabolized cocaine, which is important to note and may be a significant contributing factor to her death. With each of these, there's a concentration present. Cocaine

is broken into two metabolites: Benzoylecgonine, as well as an equaline monomethyl group, which is either anhydrous or not, so basically there's products of cocaine that exist.

Cocaine was also noted in her urine, the parent compound, but the metabolites were primarily found in her blood.

Q. We'll talk about cocaine in just a little bit. But what other drugs did you find?

A. She had Xanax, Benadryl, and the presence of a benzodiazepine, which may or may not be Xanax by itself, which is the alprazolam.

Q. So let's talk about the cocaine that you noticed from the toxicology report. Just at a real basic level, how are opiates and sedatives different from cocaine?

A. Completely. So cocaine is a stimulant. I guess Charlie Sheen would be a good example of cocaine toxicity at times. But a patient that presents with sweatiness, very elevated in their mood, very fast heartbeat, very high blood pressure, is somebody who presents with cocaine intoxication. They may or may not present with chest pain at that time.

Q. In the ER have you dealt with cocaine overdoses?

A. I see cocaine quite frequently.

Q. And so how does a patient who is undergoing cocaine overdose present?

A. Overdose, that's a very cautious word with regards to that. So a patient that can have cocaine present that is not

intoxicated, as well as be intoxicated with cocaine but not have severe life-threatening toxicity, it is a spectrum. So cocaine patients can present a wide spectrum of that. It depends on if they're in the intoxicated phase or post intoxication phase where they present with depression, you know, sedation, things like that. But an acutely intoxicated patient with cocaine is very elevated in their mood, high blood pressure, tachycardia, sweaty, they present with stimulant.

Q. Okay. And how would that compare with someone who has received too many sedatives and opiates?

A. The opposite. So patients with sedatives present with depression in their mental status, their breathing may or may not be slower, they tend to be stuporous, basically meaning that their cognition is declined. So the opposite of cocaine intoxication.

Q. And how common is cocaine-induced heart attack?

A. So, that's a very complex question. So the patients that present to the -- with cocaine chest pain, the incidents of myocardial infarction is about 6 percent. The incidents of abnormal heart rate, for instance, is about 0.14 percent.

Q. Okay. So what does that mean, that first 6 percent number?

A. The means that patients that are actively having chest pain that presented to the hospital or actually presented to the Emergency Department from the studies, only 6 percent of them had evidence of a myocardial infarction.

Q. Which is meaning?
A. Meaning heart attack.
Q. And so what about the other 94 percent?
A. They did not have heart attack.
Q. Meaning that there was some cocaine in the system, but it hadn't caused a heart attack?
A. Correct.
Q. Okay. And then the second number that you gave us?
A. So that's what is important with cocaine. So cocaine does several different things to your blood vessels and to the body. As I mentioned, all of this occurs from blocking the reuptake of the excitatory chemicals. So that makes you jazzed up. So that makes norepinephrine, dopamine, all of these chemicals higher.

The issue is, is that you have an increase in risk for spasm of your blood vessels, which is primarily the mechanisms by which cocaine causes chest pain. It also can increase the progression of plaque or that junk in the blood vessel, the atherosclerosis. And it can cause acceleration of clot formation.

Additionally, you can see patients have seizures, bleeding in their head, tearing of their blood vessels, spasm of the blood vessels in the gut. So there's a variety of spectrum of pathology that can present with cocaine.

Q. Okay. One last thing before I ask you about the

conclusions that you reached. If Ms. James had only received these medications, the oxycodone and Percocet that have been prescribed on September 26th, 2005, and not the medication that she was prescribed ten days before, would that have been enough to cause her death, in your opinion?

A. Opiates, by themselves, would be, in isolation, lower likely to produce sudden death. They can. I can't answer that with medical certainty.

Q. Okay. Based on your review of the entire file, did you reach a conclusion regarding the cause of death of Scottie Lin James to a reasonable degree of medical certainty?

A. Yes.

Q. And what was that?

A. Multiple drugs, overdose, produced her death. No single drug, including cocaine and opiates, by themselves, would just produce an isolation or death.

Q. Is that cocaine level significant?

A. You can't answer that. It's the metabolized cocaine, which is associated with ingesting cocaine. So, having the presence of the cocaine metabolite with a single number by itself, I cannot tell you would necessarily imply intoxication nor death.

The issue with cocaine, as I mentioned, is that outside of the vasospasm, it can produce abnormal heart rhythms. So in those patients that have coronary disease, with other drugs, could this produce death, particularly in Scottie Lin James?

Yes, it can.
Q. Is it -- is there any way to tell when Ms. James ingested the cocaine?
A. Not accurately.
Q. What kind of half-life does cocaine have?
A. So it depends on the mechanism and the route of injection if you snort it, shoot it or smoke it. So the parent compound, cocaine, you feel the effects of it within minutes, depending on the route of injection. The cocaine parent compound is in the blood for about 20 minutes, maybe up to two hours.
So the presence of cocaine in her urine could imply that she had a more recent ingestion. However, cocaine can just, like all of these other drugs, have postmortem redistribution. So she could have distribution of the cocaine proper. But having the parent compound, cocaine, would potentially imply a more recent injection. But you cannot say that with definitive conclusion.
Q. And I assume that the Benadryl was not a lethal substance?
A. I don't recall what the concentration was. But the problem is that both Benadryl and cocaine block sodium channels. So if you take a lethal concentration of Benadryl, it is possible to have death from the combination of cocaine and Benadryl, or even just Benadryl alone, by itself. But I don't recall the concentration. I would have to review.
Q. Okay. Let's move on to Bryan Brigner. What was his date

of death?
A. 10/2/05.
Q. Okay. And how long did he see Doctor Volkman?
A. From 7/6/05 to 9/30/05.
Q. And was his date of death two days later?
A. 10/2/05, correct.
Q. And the time of death?
A. 7:30 in the morning.
Q. What documents did you have to review regarding Mr. Brigner?
A. Partial progress notes, diagnostic tests, prescriptions, autopsy report, toxicology report and death certificate.
Q. And let's look at the last visit that Mr. Brigner made to Doctor Volkman on September 30th, 2005. What kinds of drugs did Doctor Volkman prescribe?
A. Oxycodone; Lortab, which is hydrocodone, these are both opiate pain medicines; Valium, which is a sedative; and Soma, which is another sedative.
Q. So two opiates and two sedatives; is that right?
A. Yes.
Q. And what did you find in the autopsy report regarding the circumstances of Mr. Brigner's death?
A. That he had fluid in his lungs, he vomited into his lungs, his bladder was distended and he had pill fragmentation noted.
Q. And what's bladder distention again?

A. When the smooth muscles are not contracting accurately and you get constipation of the bladder, so to speak.
Q. And would all of these four items be associated with a drug-induced death?
A. Yes.
Q. Let's look at what you found in the toxicology report relating to opiates relating to Mr. Brigner.
A. Again, same concentrations are noted, as I mentioned. The vitreous sample, it appears that oxycodone, in particular, as well as other drugs that are lipophilic, et cetera, go into the eyes slower and come out slower. It was noted that the femoral sample was present. These are in the medical ledger as reported, again, within the range associated with death, again, but these values can be artificially elevated with postmortem redistribution.
Q. In terms of the hierarchy of sample site, are femoral and vitreous number one and number two?
A. Newer literature has stated that the vitreous samples may be equivalent of the femoral. However, the concordance of agreement on that is greater than one. So either -- several things need to be taken into account, that the drugs go in the eye slower and out slower, so this may be a more observed phenomenon of postmortem redistribution than appreciated in the past.
Q. Are these sample sites better than heart blood?

A. Yes.
Q. And then how about for hydrocodone, what levels did you see from the femoral and vitreous samples taken?
A. Again, they were consistent with the medical literature that was noted for hydrocodone-associated deaths. But, again, the values in isolation cannot necessarily be seen together. They may be artificially elevated associated with postmortem redistribution.
Q. Okay. But let's -- oxycodone and hydrocodone are present. And were sedatives present, as well?
A. Yes.
Q. What's the significance of those -- the combination of opiates and sedatives being found in the blood? Just the presence, not the number.
A. The presence of those drugs, coupled with the history that we've seen in the past, again, coupled with the fluid in the lungs, vomiting into the lungs, extension of the bladder, is consistent with opiate-induced direct death, coupled with sedative hypnotics.
Q. Now, it looks like there were a few other drugs that were found in Mr. Brigner's system. Can you explain what those are?
A. The Sertraline is an antidepressant. Actually, Sertraline is cardio protective. There was a study performed after patients with myocardial infarction that found that there was no greater increase and risk of cardiac death or increase in

clots, because it interferes with the secretion of serotonin, which is one of the chemicals that it works with to prevent platelets from forming together, meaning there is no increase in risk of clot.

Q. So this would counteract some of the negative effects --

A. Potentially.

Q. -- that occur? And how about the other drugs?

A. So Haldol deserves mention. So haloperidol is a typical antipsychotic. It may produce abnormal heart rhythms or it may not. It is noted to increase the heart cycle associated with -- it is noted to associate -- to increase the electrical heart cycle, which may predispose you to abnormal heart rhythms.

Q. Now, wasn't an antipsychotic one of the other drugs that you needed to consider in prescribing opiates and sedatives?

A. Yes.

Q. And walk me through that again. Why would it have been a red flag?

A. Just because, again, the risk for drug/drug interactions.

Q. Did you consider the potential for a heart attack by Mr. Brigner?

A. Yes.

Q. Explain to us what your findings were.

A. So this was provided after the time of my report, I believe. And so he was found to have significant coronary

lesions. He had some confusing statements noted by the pathologist with regards to the autopsy. He was found to have two large plaque lesions of his right coronary artery, which is just basically the right side of the heart and the bottom part of the heart.

One of the lesions was -- I believe it was the distal lesion was found -- meaning towards the bottom of the blood vessels -- was found to have blood in it. That can be a significant marker for an unstable plaque, which may trigger acute heart attack. And I felt that that was to be noted significantly. So he does have evidence of significant coronary lesions.

There's also another lesion of the LAD. But what was confusing to me was that the pathologist noted that no evidence of acute clot was noted in the blood vessel, no evidence of acute scar or infarct was noted.

Q. And would those be things that you would expect to see from a heart attack induced death?

A. You would assume, yes. But the issue, as noted in the overall assessment of this patient, is somebody that had acute coronary lesion with multiple drugs present.

Q. And, again, what effects do opiates and sedatives have on the heart's ability to function?

A. Well, it will depress its function over time. But patients with significant coronary obstructing lesion, in this case he

had this hemorrhagic plaque, I can tell that you this was not an acute heart attack in itself. And the presence of Haldol also could have triggered an abnormal heart rhythm as oxygen would decline and his carbon dioxide would go up, which predisposes you to increased heart rhythms. So it's multifactorial.

Q. How do you approach someone who has got a heart condition in terms of deciding how much opiates and sedatives to prescribe?

A. It depends again on their duration of treatment and things like that. When we talked about tolerance and things like that, somebody with underlying significant coronary disease, if they have oxygen requirements, if they have lung disease and things like that, you would be very cautious with the utilization of combination drug therapy.

Q. Would you even prescribe that kind of slurry of medications for somebody who had a bad heart?

A. The drugs themselves, as I mentioned, overall don't interact with the heart specifically as much as with the lung and the interaction amongst the other organs themselves. I would not prescribe two sedatives and two opiates to anyone because of the risk for drug/drug interaction.

Q. Would you ever recommend that if someone asked you in your role as a director of medical toxicology?

A. No.

Q. What was your conclusion, to a reasonable degree of medical certainty, regarding the cause of death of Mr. Brigner?
A. That he had significant cardiac lesion and was found to have significant drugs present. He had evidence of combination drug overdose and significant cardiac lesions. I cannot describe a specific cause of death.
Q. So it wasn't one or the other, from what you could tell?
A. I could not weigh what was a greater likelihood. They were both present. He had significant lesion present.
Q. If the pathologist explained what he or she meant, would that potentially change your opinion?
A. Potentially. But, as I mentioned, that hemorrhagic plaque is a significant risk factor for acute myocardial infarction. So I still think that's a significant value.
Q. Let's move to Ernest Ratcliff, who died on October 23rd, 2005.
What was the date of his first and last visit with Doctor Volkman?
A. 10/21/05, from the chart.
Q. And how many days after that visit did Mr. Ratcliff die?
A. Two.
Q. And how old was he?
A. Thirty-eight.
Q. And his time of death?
A. Eight a.m.

Q. What sources of information did you have relating to Mr. Ratcliff?
A. Progress note, prescriptions, toxicology report and death certificate.
Q. By progress note, do you mean singular, just one progress note?
A. I would have to -- well, I mean, it was the note that was from 10/21.
Q. Let's look at the one prescription that Mr. Ratcliff receives from Doctor Volkman. What kinds of drugs do we see here?
A. Same drugs that we've seen in the past prescribed, which are oxycodone, Lortab, which are two opiates, and two sedatives, which are Xanax and Soma.
Q. And what did we see -- actually, let's -- one other thing that we haven't talked about yet. We've noted a few times individuals who have concerns regarding compliance, and I was wondering, as a toxicologist and as a physician, your view on giving someone a prescription for 660 pills at one time, if there are compliance concerns.
Would that be something that you would do?
A. That would be very concerning.
Q. And why would that be?
A. Because, again, the risk for adverse outcome. That is a large quantity of medications.

Q. Well, what would you worry they would do?
A. You have the question of risk for overdose; you have the risk for, again, if they're noncompliant, could they be dispensed to other individuals and not themselves. So those would be my concerns.
Q. And have you ever had those concerns in dealing with a patient?
A. For?
Q. In terms of prescribing medication.
A. Yes.
Q. And what have you done as a result of those concerns?
A. If I am concerned that the patient would have either diversion of those agents or risk of significant toxicity, I do not prescribe those agents.
Q. Let's look at what you found in the toxicology report relating to Mr. Ratcliff's death. What drugs were present?
A. Oxycodone, hydrocodone and methadone.
Q. Are these all three opiates?
A. Yes.
Q. And the oxycodone and hydrocodone was obviously prescribed by Doctor Volkman. And the methadone was not, correct?
A. Correct, there was no prescription for methadone.
Q. Let's go through each one on its own. What about the level of oxycodone?
A. It is noted it is in the range but, again, there was no

sample site. The drug itself undergoes postmortem redistribution. So the value report is there. Is it within the range? Yes. Could it be artificially elevated? Yes. So it is noted.

Q. And how about hydrocodone?

A. Same thing. It was noted. It is .07, it was slightly below the range. But as we mentioned, it can undergo postmortem redistribution, it can be artificially high. The combination of agents may have lower values cumulatively when there are multiple drugs present.

Q. And for the ranges, are those just single drugs associated with death?

A. Yes.

Q. And the third one is methadone. What was the significance of that?

A. Methadone is very significant, and I would state that that is perhaps one of the most important findings out of this.

Q. And why is that?

A. Because methadone by itself has significant lethality. The reason being is that methadone's half-life is up to 54 hours. And oftentimes what we see is that when patients have a single ingestion, particularly even if they're tolerant individuals, they have a high risk of drug death for a variety of reasons. Number one is the delayed onset of absorption, which is what we talked about in the past, of unexpected absorption in blood

levels. So they have an apparent sudden onset of respiratory death.

The other issue too is that methadone interferes with a specific channel in the heart which creates abnormal heart rhythms. So it is lethal by two mechanisms.

Q. And what was your -- and there's also alprazolam present. What is that?

A. Xanax, the sedative.

Q. So what was your conclusion to a reasonable degree of medical certainty about what caused the death of Mr. Ratcliff?

A. Multi-drug death.

Q. And could you say whether it was the oxycodone, or the methadone, or the hydrocodone, or the Xanax?

A. As I mentioned, you cannot ascribe to any single agent with regard to -- they're all opiates. My concern is that the interpretation of that methadone value, no one can accurately do. Methadone is a significant component. And I would place a greater concern on its presence. So I would not ascribe it to any single agent, but I think methadone is a significant component associated with his death.

Q. And could you say that he had died -- if it was only methadone, would you be able to say that that would have caused his death?

A. The drug concentration itself, again, in isolation doesn't. But if he had clinical toxidrome of fluid in his lungs, et

cetera, but the presence of methadone with an unexpected death could absolutely imply drug death. Methadone is a significant component and risk factor for a drug death by itself.
Q. And if it was only oxycodone and hydrocodone, would that have been sufficient to cause his death?
A. In a clinical toxidrome, present with fluid in his lungs and things like that, given the prior described history that we've talked about.
Q. Mark Reeder, November 19th, 2005, date of death. How long did Mr. Reeder -- or when did he receive his last prescription from Doctor Volkman?
A. 10/25/05.
Q. And how about when did he -- how long after that did he die?
A. On 11/19/05, which I believe is like 25 or something days.
Q. His age?
A. Thirty-four years old.
Q. And time of death?
A. 10:40 a.m.
Q. And what information did you have to review regarding the death of Mr. Reeder?
A. Progress notes, prescriptions, diagnostic studies, autopsy, toxicology report and death certificate.
Q. What warning signs existed relating to Mr. Reeder?
A. He had significant morbid obesity and was a smoker.

Q. And obesity and smoking is a concern for these drugs because of what reason?
A. Because of the respiratory variance that we spoke about.
Q. Let's look at the prescription that Mr. Reeder received on his last visit to Doctor Volkman. What drugs was he prescribed?
A. Oxycodone, Norco, which is hydrocodone, so two opiates, and Xanax and Soma, which is two sedatives.
Q. Taking the oxycodone, 30 milligrams, ten pills a day, is that within the half-life of oxycodone or is it one of these situations where it is going to be overlapping?
A. It would be overlapping if the time interval is less than the 2.5 to 6 hour half-life.
Q. Okay. So is there a way that Mr. Reeder could have taken these while not tripping over the half-life of oxycodone?
A. Yes.
Q. How would that have had to work?
A. So he would have to take, for example, two to three tablets every six hours, or greater.
Q. And is it possible to do that while you're also taking Norco, Xanax and Soma?
A. It's possible to take those two. But, again, the combination of multiple opiates with multiple sedatives, again are centrally acting agents, which dramatically increases your risk for adverse breathing, irrespective of the initial dose

interval for one of these agents.
Q. So is there a safe way he could have taken all four of these medications?
A. In my opinion, no.
Q. Let's look at the toxicology report for Mr. Reeder. What did you see?
A. The presence of the oxycodone and hydrocodone.
Q. And I see that the hydrocodone is below the range of levels associated with death?
A. Again, as we noted, these values are present. They may be artificially elevated with postmortem redistribution. The concentrations that I had listed were associated with hydrocodone deaths, so it is below.
Q. And then what other drugs were present in the system?
A. Carisoprodol, which is Soma; alprazolam, which is Xanax; and alcohol, which is another depressant.
Q. Oxycodone, hydrocodone, carisoprodol and alprazolam were all prescribed by Doctor Volkman; is that correct?
A. Yes.
Q. And the range level for hydrocodone is only dealing with a one-drug situation as opposed to a four-drug situation; is that right?
A. Correct.
Q. Was the alcohol significant at all in your read?
A. It is another depressant, so it may be a significant

contributing factor.
Q. Did you consider the possibility of heart death relating to Mr. Reeder?
A. Yes.
Q. And what did you conclude?
A. Again, notation that heart disease existed. He had a blockage of his blood vessel, but no clot was seen, no plaque hemorrhage was noted, there was no infarct or scar noted on the gross heart or on microscope.
Q. So none of the indications that you expect for a heart-induced death?
A. In my opinion, yes.
Q. Meaning that --
A. There was no other notation of the findings for myocardial infarction.
Q. And what was your conclusion to a reasonable degree of medical certainty regarding cause of death of Mark Reeder?
A. That he died as a result of multiple drug overdose.
Q. A few more charts.
MR. WRIGHT: But may I have a moment, Your Honor, before I wrap up?
THE COURT: You may.
Do you need some water?
THE WITNESS: Yes, ma'am, if you wouldn't mind.
THE COURT: Ms. Brown, I think maybe a fresh cup would

be a good thing.

(Courtroom deputy complied.)

BY MR. WRIGHT:

Q. One thing before we wrap up. We talked a little bit about blood levels earlier on, including the reports that you saw in the medical records.

Do you remember that?

A. Yes.

Q. Now, that level is different than the blood levels that we were talking about in terms of the toxicology reports, right?

A. Drug read levels reported from the laboratory values, for example, meprobamate or the urine levels, is that what you're speaking of?

Q. I'm talking about is there a difference between blood levels for someone who is alive versus blood levels for someone who is dead?

A. Yes.

Q. And for someone who is alive, can you use blood levels to determine compliance with a particular dosing regimen?

A. Can you determine compliance for when you're alive with dosing regimen? For a single drug, it is possible. For multiple drugs, it's complex and difficult. But to compare a living blood sample value to a postmortem value sample to determine toxicity is incorrect.

Q. Why is that?

A. Because the volume of distribution of drugs after death is different. Therefore, a plasma concentration is not equivalent. So it is a specific formula to determine what is concentration, plasma -- what is a blood level achieved. So when they're measuring them, these are in people that had died and saying, these are what these values exist, they may be elevated, they may be low. But to ascribe a living blood sample value of toxicity to a postmortem value of toxicity is incorrect.

Q. I would like to walk through just looking backwards at the twelve individuals that we've reviewed. And the first thing is this chart entitled Combination Of Drugs Prescribed. What does this tell us, Doctor?

A. That of the twelve patients that are listed here, 100 percent of them have a combination of opiates and sedatives.

Q. And at a real basic level, what does that combination -- what concerns does that kind of combination raise?

A. Combination of two centrally acting agents increases your risk for adverse outcome as it applies to breathing.

Q. And how about the numbers that received two opiates and two sedatives?

A. 75 percent of the patients, basically nine of the twelve, received two opiates and two sedatives.

Q. Now, let's just look at the two sedatives. What

synergistic effects exist when you prescribe Xanax and Soma?
A. It is exactly that, it is synergistic, it increases the likelihood of adverse outcome. But sedatives alone have a higher therapeutic index of safety, meaning that these drugs are harder to die of as a result of overdose or taking them. But when you combine them, that safety factor decreases.
Q. And in your experience, have you ever prescribed two opiates and two sedatives at the same time?
A. No.
Q. And if anyone asks you if they should, what would you tell them?
A. No.
Q. Let's look at the combination of drugs that these individuals received at the time of their death. What does this show us?
A. That of the patients listed there, you can see the proportionality of prescriptions of oxycodone, hydrocodone, benzodiazepines, which were either Xanax or Valium, and Soma, which is the other sedative.
Q. So it looks like everyone received the benzodiazepines, correct?
A. Correct.
Q. And then 10 out of 12 received Soma?
A. Correct.
Q. And is Soma a drug that you've ever prescribed?

A. No.
Q. And what are the two Xs for oxycodone? I see that for Mr. Gillespie, Mr. Jordan and Ms. James.
A. Those were two prescriptions for oxycodone.
Q. So it looks like, is it right that eleven out of twelve received some form of oxycodone; is that right?
A. Yes.
Q. Now, let's look at the ones who received the exact same combination.
A. 50 percent of the patients that are presented here received the exact same prescription of oxycodone, hydrocodone, benzodiazepine, which was Xanax, and Soma.
Q. Now, you mentioned earlier on that there were a number of patients' specific considerations that you needed to take into account when prescribing medications.
Do you remember that?
A. Yes.
Q. Is this kind of pattern consistent with those kinds of patient specific concerns?
A. This would not appear to take into account patient specific drug tailored therapy.
Q. So what about something like obesity, how would that change the kinds of drugs that someone would receive?
A. Again, so those patients that have significant obesity are a greater risk for respiratory adverse affects by themselves,

just with the opiates by themselves.

The combination use of a sedative and an opiate in an obese patient puts them at greater risk for central respiratory depression.

Q. What do you do when someone is obese and they're asking you for these medication?

A. So you have to weigh what is the most appropriate therapy. Muscle relaxants -- or I'm sorry, sedatives are not analgesics, they're not pain relief, per se. So the question is: What is the most appropriate patient-tailored therapy?

Q. And in your experience, would an individual like Danny Coffee, Mary Carver, James Estep, Dwight Parsons, Ernest Ratcliff and Mark Reeder receive the exact same combination of drugs?

A. Would I prescribe the same drugs or is there concern that they were prescribed all the same drugs? Yes, there is concern.

Q. Now, let's look at the date of the last visit to Doctor Volkman compared to the date of the death. And I am just going to skip ahead to the next slide. What does this show?

A. So all of these patients were patients that received the exact same combination drug therapy, which was oxycodone, hydrocodone, Soma and Xanax. And they all, at the time of their death, died within a significant short period of time from receiving oftentimes a change in that medication dose just

prior to their death.
Q. So 11 out of 12 died within five days of their visit; is that right?
A. Yes.
Q. How many died within two or one day of their visit?
A. Eight.
Q. And how many of that group of 12 received an increase in the prescription in their last visit to Doctor Volkman?
A. Eleven of the 12.
Q. And why would that be significant?
A. Again, when you accelerate the dosing of these medications, again, that particular combination of sedative hypnotics -- I'm sorry, sedatives and opiates, that safety index declines.
Q. The safety index, explain to me how that impacts your decision whether or not to increase the medication someone is already receiving.
A. So, as we mentioned with regards to the tolerance, you can become tolerant to the pain relief effects. But the tolerance to the side effect profile is still limited by that adverse effect profile.
So as you accelerate the dosing regimen and frequency of these drugs, particularly two centrally acting agents, the risk of harm dramatically increases.
Q. Now, you said two centrally acting agents; what do you mean?

A. They both act in the same synergistic way, they both depress respirations and cause sedation.
Q. Now, when you say two centrally acting medications, are you talking about --
A. Opiates and sedatives.
Q. Okay. So you're talking about the nine out of twelve who are receiving two opiates and two sedatives?
A. Yes.
Q. Would the same concern apply for someone who is receiving an opiate and a sedative?
A. Yes.
Q. Because -- okay, let's look at the age of death and I'll just skip ahead. How many were under the age of 40?
A. 75 percent.
Q. And then that isn't counting Mr. Jordan, who was 40 years old; is that right?
A. No.
Q. Is this the kind of list of ages that you would expect to be associated with heart attack induced death?
A. It would be lower likely. This is the same epidemiology characteristics of patients that have unintentional drug overdose death -- they tend to be under the age of 50, 40, they tend to be younger and, again, have multiple drugs present.
Q. And is that the kind of situation that, you know, when you're at Good Sam on a Saturday night that you see something

coming in, this profile of a person?
A. This is the profile of the patients that I see that present with combination drug therapy overdoses.
Q. And I just wanted to ask you about the time of death. How many died in the morning?
A. 66 percent.
Q. And why is that significant?
A. Again, we talked about the respiratory variance that occurred nocturnally, or at night. So chronic opioid therapy, by itself, changes that pattern of breathing at night. Those patients that are obese, that are now on opiates themselves, that have combination drugs, are at significant risk for respiratory depression, particularly at night.
Q. And that's because your breathing pattern changes when you sleep?
A. Yes.
MR. WRIGHT: No further questions, Your Honor.
MS. CROSS: Your Honor, may we approach?
THE COURT: You may.
(The following transpired at a sidebar conference.)
MS. CROSS: Your Honor, I'm trying to abide by the schedule, but I have to use the restroom and I don't know if it would be more appropriate to go take the break now or I can just press on until 3:00.
THE COURT: Okay. Well, it is not going to cause a

problem, I don't think, for anybody other than you if we don't take the break, so we will.

MS. CROSS: Thank you.

(Ends sidebar conference.)

THE COURT: All right. Folks, we're going to take the afternoon break early, from now until 2:45. Let me just remind everyone not to discuss the case among yourselves or with anyone else or permit anyone to discuss it with you or in your presence. Report any violation to Ms. Brown. No research or investigation over the break on your own regarding the case or any issue or person involved. And no Internet chitchat of any kind regarding the trial.

See you back in 15 minutes.

(The jury left the courtroom at 2:33 p.m.)

THE COURT: Doctor, you can take a 15-minute break now, and no discussion of your testimony with anyone.

THE WITNESS: Yes, ma'am.

THE COURT: Counsels, anything you would like to put on the record in the absence of the jury before we take a break?

MR. WRIGHT: No, Your Honor.

MS. CROUSE: No, Your Honor.

THE COURT: Okay. See you back in 15 minutes.

(Recess.)

(The jury entered the courtroom at 2:59 p.m.)

(Doctor Policastro resumed the witness stand.)

A JUROR: Your Honor, if I may, my apologies to the Court for delaying the proceedings. A business call interfered, and it put me over a couple minutes and therefore it delayed the jury from promptly returning.

THE COURT: Okay. Apology accepted. Thank you.

CROSS-EXAMINATION

BY MS. CROSS:

Q. Good afternoon, Doctor Policastro. Your field expertise is in the area of toxicology and emergency medicine?

A. Yes, ma'am.

Q. And toxicology is basically the -- simplified -- is the study and evaluation of the adverse effects of chemicals or drugs?

A. Yes.

Q. And toxicology is an interdisciplinary science, right? It integrates fields of other areas of medicine, right?

A. Yes.

Q. It integrates the principles and methods of chemistry?

A. Yes.

Q. Biology?

A. Yes.

Q. Pharmacology?

A. Yes.

Q. Physiology?

A. Yes.
Q. And medicine, right?
A. Yes.
Q. But not in that list is pathology, right?
A. No.
Q. You are not trained in pathology?
A. No.
Q. You have no experience in pathology?
A. I took pathology as a training in medical school. It was not a specific class. I did not have a fellowship nor residency in pathology.
Q. So you have no experience in pathology, correct?
A. No.
Q. You are not a coroner?
A. No.
Q. Never have written an autopsy report?
A. No.
Q. Never performed an autopsy?
A. No.
Q. That's what pathologists do, correct?
A. Yes.
Q. And pathologists not only perform autopsies, which not only determine cause of death, but they also may discover information about genetic progression of diseases, correct?
A. Yes.

Q. Now, determining cause of death involves much more than merely determining a person's blood concentration level, correct?
A. Yes.
Q. Now, you were asked to give an opinion about the effects of certain drugs in this case, right?
A. Yes.
Q. And your opinion today is that the drug interactions in the cases that you reviewed could and did result in respiratory depression and death?
A. Yes.
Q. Now, if a pathologist were to disagree with you on your conclusions, you would just take issue, right?
MR. WRIGHT: Objection.
THE COURT: Sustained.
Q. Now, as an emergency medicine doctor, an ER doctor -- can I say it like that?
A. Yes.
Q. You've never seen a patient more than one time, have you?
A. That is not correct.
Q. Well, you don't treat patients in the ER over long periods of time, correct?
A. Correct.
Q. You don't take care of continuing chronic problems in the ER, correct?

A. Incorrect.
Q. So in the ER, it is your testimony that you care for chronic pain patients over a long period of time?
A. No.
Q. Now, your resume -- and you've testified that you are board certified in emergency medicine, right?
A. Yes.
Q. And clinical toxicology or medical toxicology?
A. Yes.
Q. And we say medical or clinical toxicology because there are all different types of branches of toxicology, isn't there?
A. Yes.
Q. But your specific area is clinical/medical toxicology, right?
A. Yes.
Q. And that involves interpreting drug concentrations, which is used to treat and give prognosis of patients, right?
A. Yes.
Q. In other words, you don't, on a regular basis, deal with dead people?
A. No.
Q. And as a clinical medical toxicologist -- well, let me ask this: There are various types of or parts of toxicology; the study of toxicology, right?
A. Yes.

Q. And forensic toxicology is one of those parts?
A. Yes.
Q. And you are not board certified in forensic toxicology, are you?
A. No.
Q. You are not a member of the American Board of Forensic Toxicology?
A. No.
Q. Nor are you a member of the American College of Forensic Toxicology, right?
A. No.
Q. In the field of forensic toxicology, and I know your area is medical clinical toxicology, but in the field of forensic toxicology, there is a branch of it or a field of forensic toxicology that deals with postmortem toxicology, right?
A. Yes.
Q. And postmortem toxicology, under the umbrella of forensic toxicology, involves interpreting postmortem drug concentrations to determine whether the drugs had an effect on cause of death, right?
A. Yes.
Q. And you would agree that you have had no specific training or experience in the field of forensic toxicology, right?
A. No.
Q. So you've been trained and your experience is as a treating

physician who deals with the effects of certain drugs on living people, primarily?
A. Yes.
Q. And because you're not a pathologist or involved in forensic toxicology, you can't say what value is placed on a toxicology report by a pathologist or medical examiner, correct?
A. No.
Q. Now, we're going to get into the specifics, but I want to ask you an overall question for my understanding. Is it your testimony that if a person is on any of the combination of drugs that we've seen in the Power Point and they died, that the drugs caused their death? Is that your testimony?
A. Is any person that's on those drugs --
Q. And they die.
A. Is that the only cause of their death?
Q. Would you attribute the drugs to the cause of death?
A. No.
Q. Are you saying that in this case, though -- I guess I didn't ask my question very artfully.
My question is: In this case, given the combinations of drugs, is it your testimony that because of the combination of drugs, that the person -- the cause of death are the drugs?
A. The combination of the drugs, coupled with their co-morbid factors, yes.

Q. And is it your testimony that if a patient is on any of those combination of drugs and they don't die, that they're soon going to die?
A. Not soon going to die. They have an increase in risk to have adverse respiratory effects.
Q. So, in other words, if they're on this combination of drugs, they're going to die, is that what you're saying?
A. No.
Q. So the combination of the drugs that we've seen, you're not saying necessarily has to cause death, right?
A. Correct.
Q. Now, just asking a few questions about your education, I know that you have -- you testified that you graduated from Wright State University School of Medicine?
A. Yes, ma'am.
Q. And that was in 2001?
A. Yes.
Q. So that was ten years ago?
A. Yes.
Q. And you completed a fellowship, right, after that?
A. I completed residency first and then a fellowship.
Q. And you weren't an attending or treating physician while you were completing your fellowship, were you?
A. I was an attending physician.
Q. You was? Where? I'm sorry.

A. Where was I an attending physician?
Q. Yes, sir.
A. At Jewish Hospital and at University of Cincinnati.
Q. Okay. And so you've been an attending treating physician for how long?
A. Since I graduated from my residency.
Q. Which was in?
A. I'm sorry. I blanked out, I apologize. 2004.
Q. So you actually have been a treating attending physician for almost five years, if my math is right?
A. Yes, ma'am.
Q. And you said that your fellowship consisted of telephone consults regarding drug interactions?
A. As well as bedside consultations, correct.
Q. As well as bedside consultations. And bedside consultations in a hospital setting?
A. Yes.
Q. And were those bedside consultations in the hospital setting for chronic pain patients?
A. There was no differentiation of the patients. They were for whatever was the suspected overdose.
Q. So you can't say?
A. Every patient that I've seen may or may not have been a chronic pain patient. They were on a variety of toxins.
Q. Understood. But my question is: How many of those

patients that you saw at bedside consultation were chronic pain patients, if you know?
A. I can't give you a number.
Q. Speaking of chronic pain, you are not a pain management doctor, correct?
A. Correct.
Q. You received no training in pain management, correct?
A. Correct.
Q. And no education in pain management, right?
A. Correct.
Q. Have you even been to a seminar regarding pain management or chronic pain issues?
A. I have been to seminars dealing with the toxicology of opiate management and sedative management, and that would be inclusive of patients that have chronic pain.
Q. And you're not board certified in pain management, correct?
A. No.
Q. But you are board certified in emergency medicine?
A. And medical toxicology.
Q. And medical toxicology. Can you just describe what that means, board certified?
A. It means I completed a residency in emergency medicine. I took an oral board and written board. I'm a practicing emergency physician with that.
The medical toxicology, I took a written board.

Q. And in the medical field, being board certified is high honors, right?
A. I would say it is distinguished, yes.
Q. Distinguished honors. I mean, if your -- most hospitals and practice groups want someone who is board certified in a field, correct?
A. Correct.
Q. Because it means a lot, right?
A. It means something.
Q. Well, you've done extra training, you've done extra testing to be above those who haven't, correct?
A. Correct.
Q. But in some -- all of your employment and your experience has pretty much been in emergency medicine and medical toxicology, right?
A. Yes, ma'am.
Q. You stated that just last week you had someone in the ER who overdosed?
A. Correct.
Q. Was that overdose due to oxycodone or hydrocodone?
A. It was a combination of oxycodone, heroin and Xanax.
Q. You stated that you were -- you've been published. Can you tell the jury what that means?
A. Well, I published a textbook article on insulin sulfonylurea toxicity. So I published those textbook chapters

when I was a medical student with an attending toxicologist. I have also written articles on urinary obstruction, which was published. I am currently writing manuscripts with several other physicians.

Q. On what topic?

A. On what topic?

Q. Yes, sir.

A. Toxicology.

Q. Have you ever written any articles about chronic pain issues or pain patients?

A. No.

Q. Now, you mentioned -- I believe we'll get into it a little bit in more detail, but I believe you mentioned that there was a publication that you relied on in forming your conclusion. I'm not saying the only, but you relied on it, Baselt?

A. Baselt is a textbook that was one of many resources that I looked at.

Q. And in looking at that textbook for the support of your conclusions, you were looking at ranges of therapeutic and toxic levels of drugs?

A. I was utilizing that, amongst several articles, correct.

Q. And when you looked at that Baselt, B-a-s-e-l-t, I may not be pronouncing it correctly, textbook, Baselt actually describes ranges, therapeutic and toxic ranges of drugs for opiate-naive people, correct?

A. That's why I mentioned the articles that I looked at.
Q. But is that correct?
A. It is representative of a variety of patients. Baselt was the least important resource that I used.
Q. I didn't ask you how much value you placed on it yet, Doctor Policastro. I just asked you simply does Baselt give you ranges for therapeutic and toxic levels for people that are opiate-naive?
A. I would have to review the resource again.
Q. So you don't remember?
A. I do not recall specifically. Baselt accumulates other articles and reports the values that is reflected in those articles. I would have to relook at every specific article that it mentioned in that.
Q. But you would agree with me that Baselt does not deal with therapeutic ranges for chronic pain patients, right?
A. I would have to relook at the article as it relates to those values.
Q. Are you saying you don't remember?
A. I'm stating I do not recall.
Q. But it is a resource that you relied upon in forming your conclusions here?
A. It was one of 34 resources that I looked at. I do not recall the exact specifics of the articles that they mentioned in the reference range.

Q. So is that yes to my question?
A. Is what yes?
Q. You mentioned a medical phenomenon, for lack of a better word, qualitative synergistic effect. You've said it quite -- numerous times here.
What does that mean?
A. It means that drugs that are present that act together.
Q. That's it?
A. It means exactly that.
Q. Okay. And the qualitative synergistic effect was the most important factor in your conclusions that you spoke about earlier, right?
A. Given the other co-morbid factors, yes.
Q. My question is it was the most important factor that you used, that you considered, right?
A. Yes.
Q. Now, if I were to look up in one of the textbooks that you mentioned, one of the 34 textbooks or resources, I'm sorry, that you said you relied on to give your opinion in this case, where would I find the qualitative synergistic effect phenomenon?
A. It is not specifically mentioned in those terms.
Q. So I would not be able to find it, correct?
A. You would be able to find evidence listed that combination of multiple central acting agents is a significant risk factor

for overdose and respiratory depression.
Q. So -- so qualitative synergistic effect, as you termed it, would not be something that we might hear from another medical personnel that came in here?
A. I can't speak to what other personnel would state to.
Q. Is it something commonly referred to in your practice, qualitative synergistic effect?
A. As a toxicologist, yes.
Q. So other toxicologists speak in those terms as well?
A. Correct, the differentiation between a number and the drug proper.
Q. And do toxicologists oftentimes work in conjunction with pathologists?
A. Are you speaking about medical toxicologists or forensic toxicologists?
Q. Either.
A. I can't speak who they work in conjunction with. There's meetings with, we have conversations with.
Q. As a medical toxicologist, do you work in conjunction with pathologists?
A. On a routine basis, no.
Q. And so is qualitative synergistic effect a term that a pathologist would readily hear?
A. I can't speak to what a pathologist would regularly hear. I'm not a pathologist.

Q. Now, I believe you stated that this was your first court appearance where you've been qualified as an expert, right?
A. Yes, ma'am.
Q. And how many times have you been consulted for expert testimony in a court case?
A. How many times total?
Q. Yes, sir.
A. Four.
Q. And have they all been on behalf of the government?
A. No.
Q. Have they been civil cases?
A. Two were.
Q. So two cases were civil and two were criminal?
A. Correct.
Q. Now, Doctor, you would agree with me that untreated pain is a serious problem in our country, correct?
A. Yes.
Q. In fact, experts agree that tens of millions of Americans suffer from either undertreated or untreated pain, right?
A. I don't know where you're getting that statistic, but, yes, people are in pain that have not been treated.
Q. And -- well, I'm not really saying a statistic, but I'm saying that tens of millions -- do you know that tens of millions of people, Americans, suffer from either undertreated or untreated pain? Have you even heard that before?

A. I've heard that people have undertreated pain.
Q. Have you heard of those numbers before?
A. I would have to see your resource. I'm not exactly sure where you're using that from.
Q. I'm just asking you, have you heard that?
A. I don't know your statement, where it was qualified in a medical journal. Are people treated that have untreated pain? Yes.
Q. Well, let me ask this: Have you heard of the American Pain Foundation?
A. I'm not familiar with it.
Q. You don't know that that's a professional organization of pain specialists?
A. I'm not a pain specialist.
Q. Well, let me ask this: The medical field of treating chronic pain was fairly in its infancy back in 2005, correct?
A. I can't speak to what the field was in its infancy.
Q. Because you don't know about chronic pain or pain management?
A. I am not a pain physician.
Q. So you're not really here today to speak on opiate therapy then, opioid therapy then, correct?
A. No.
Q. Because you can't; you're not trained or educated in that field?

A. Correct.
Q. Now, I believe you stated -- correct me if I'm wrong -- that you have treated chronic pain patients in the ER?
A. Chronic pain patients presenting to the emergency department, yes.
Q. And you've treated them?
A. Yes.
Q. Isn't it true that the American Society For Pain Management recommends that when treating chronic pain patients, the dose of opiate should be increased by between 50 and 100 percent at each visit until the pain is alleviated?
A. You're speaking of an outpatient therapy that I cannot speak to, nor an organization that I'm a member of.
Q. Because ER is not an outpatient -- isn't ER an outpatient facility?
A. It's connected to a hospital. Depending upon where I work, a large percent of those patients will be admitted to the hospital.
Q. But most people come to the ER, get patched up and they're out, right?
A. I would not agree with that.
Q. Okay. Well, some people come to the ER, get patched up and they're out, right?
A. A certain percentage of those patients, correct.
Q. Okay. And so my statement about the recommendation for

increasing dosages of opiate levels, you said you would not know about because I'm speaking about an outpatient?
A. By what -- the statement that you made, you were talking about outpatient chronic pain management.
Q. What's the difference between outpatient chronic pain management and where you are?
A. You referred to a continuing of care, an acceleration of that care. Where I practice would be acute management of that chronic pain. If I'm making significant adjustments to those medications and if they had a pain treating physician, I would contact them.
Q. What if they don't?
A. Well, then who is prescribing their chronic pain medicine? Some physician is. And I would contact that physician.
Q. When you looked at the recommended dosages for and schedules of drugs that Doctor Volkman prescribed in this case -- you did do that, right?
A. Did I look at his prescriptive dosing? Yes.
Q. Okay. And where did you research to determine the recommended dosages and schedules for the drugs involved in this case?
A. I didn't research specifically the recommended dosages. It is part -- it was part of my clinical training, as well as within the resources with regards to the kinetic data of the drugs.

Q. Okay. Slow down because you lost me here. So when you looked at the dosages and the various drugs involved in this case --
A. Yes.
Q. -- you're saying that you never researched what the recommended dosages were for those types of drugs?
A. Your statement is confusing to me. What I relied upon was what is the pharmacology action of those drugs. Irrespective of the dose proper, there's a specific kinetic data of those drugs.
Q. So -- and I'm not trying to put words in your mouth. Does that mean that you didn't consider nor was it important what the recommended dosages and schedules were for the drugs that you evaluated in this case?
A. It is important. But you asked me did I consult a specific single resource to determine what that dosing schedule is.
Q. And you did not?
A. I utilized all of the resources that were noted in my report, plus my clinical training.
Q. You would agree that chronic pain can be brought on by a wide variety of illnesses, correct?
A. Yes.
Q. Like cancer?
A. Yes.
Q. Low back disorder?

A. Yes.
Q. Rheumatoid arthritis?
A. Yes.
Q. Shingles?
A. Yes.
Q. Post-surgical pain?
A. A variety of conditions, yes.
Q. Trauma?
A. Yes.
Q. Fibromyalgia?
A. There is significant debate if fibromyalgia is a significant disease per se, but it is an observed rheumatologic phenomenon.
Q. A lot of various illnesses can cause chronic pain, correct?
A. Yes.
Q. And again, you would agree that chronic pain is undertreated?
A. Chronic pain exists. It should be treated. I don't know if it is overtreated or undertreated. I can't affirm that statement nor deny it.
Q. Why not?
A. What is your source from you stating that chronic pain is undertreated?
Q. So you're saying that you just can't make a determination whether it is undertreated or untreated in this country?

A. Correct. It's treated, it's undertreated -- patients have pain, patients need to be treated for pain. I don't know the proportionality, incidence or prevalence of chronic pain that's treated or untreated.
Q. What are some of the common physical characteristics of a person who may have unrelieved pain? Can you describe that for the jury?
A. Patients that have un--
Q. Unrelieved pain. What are their physical characteristics?
A. Variance upon the patient.
Q. What are some of the characteristics?
A. There may be grimacing, there may be posturing. They may be laying supine, they may be agitated, hostile, they may be flat in their affect. They may have a variety of conditions that would present that way.
Q. Can they also appear to be a drug seeker?
A. Patients could have any appearance. I wouldn't necessarily describe what you stated.
Q. But one of the characteristics could be that they appear to be a drug seeker, right?
A. Yes.
Q. And someone who has unrelieved pain who may appear to be a drug seeker could potentially look like a drug addict to a nonmedical person, right?
A. I can't speak to what they would look like to a nonmedical

person.
Q. Could they look like a drug addict to a medical person?
A. Anyone could look like anything to anyone.
Q. Now, we're going to get to the slide in a minute, but you testified about a number of factors that would concern you in prescribing pain narcotic medicine.
Do you remember?
A. Yes.
Q. And it's not against the law or inappropriate to prescribe pain medication to a pain patient who has had a history of drug abuse, is it?
A. No.
Q. It is not against the law or necessarily inappropriate to prescribe pain medication to someone who is obese, correct?
A. No.
Q. And I'm not going to go through all the other factors that you talked about that concern you, but my point is, it is a case-by-case determination, right?
A. Correct. There should be caution in prescribing medications to patients that have specific disease states.
Q. So you're not -- you didn't testify to the jury that if a patient has some of these physical attributes that would concern you that you wouldn't treat them?
A. No.
Q. You would treat them for their pain, correct?

A. Correct.
Q. Despite obesity, right?
A. Correct.
Q. Or drug history?
A. Correct.
Q. Or any of the other factors you've mentioned, right?
A. Correct.
Q. What about heart disease? If a patient presented and they were an established chronic pain patient and they had a history of heart disease, would you not prescribe pain medication for their pain?
A. There's a double negative there. I would prescribe pain medication.
Q. Okay. I'm sorry, I didn't mean to be confusing.
Can we talk about dependence versus addiction for a moment? There is a difference, correct --
A. Yes.
Q. -- between physical dependence and addiction, right?
A. Yes.
Q. And a patient who is incapacitated by pain will necessarily become dependent on their pain medication, right?
A. It's possible.
Q. If a person's in pain and the pain medicine gives them relief, then their body will become dependent on that medication to provide relief, right?

A. Other modalities can be used for pain relief other than pain medication.
Q. I understand, but I'm only talking about pain medication.
A. Yes.
Q. And that's quite different from addiction, though, right?
A. Correct.
Q. Can you describe for the jury the difference?
A. What is the difference between addiction and physical dependence?
Q. Yes, sir.
A. Addiction would be individuals that attempt to make mechanisms by which to continue to achieve a specific agent. I mean, anything can be an addiction per se. So I am not --
Q. Fair enough. With regard to pain medication, addicts will take some of the drugs that we've seen in this case for a euphoria, a euphoric feeling, to get high, right?
A. Maybe or maybe not. They will also try to prevent the physical withdrawal of that agent. So they may not have a euphoric feeling with it. It is to prevent, as you mentioned before, a physical dependence.
Q. But the difference between the addict and the pain patient, the established pain patient, is that the established pain patient doesn't get the euphoric high from the medication; is that right?
A. I would not state that.

Q. So your testimony is that they do get high off of their pain medication that they're dependent upon?
A. I'm not suggesting that they may or may not. A level of euphoria is not necessarily different between someone that's a chronic pain management patient and an addict, per se.
Q. So if a person is a chronic -- established chronic pain patient and they have built up tolerance for their medication, do they still get the high or the euphoria, is that your testimony?
A. They may or may not. I can't state definitively. Just because you have a tolerance to sedation doesn't mean that you don't have euphoria. Just because you have a tolerance and require an increasing dose does not mean that there's not an associated euphoria.
Q. So there could be euphoria?
A. Yes.
Q. Now, help me with the testimony that you said -- I believe you said there's no tolerance to respiratory depression, correct?
A. No, I did not say that.
Q. You did not say that?
A. There is tolerance to respiratory depression after a certain period of time.
Q. Okay.
A. I don't believe I ever said that there's no tolerance to

respiratory depression.
Q. That's fair, I may have written it down wrong. But your testimony is that there is tolerance to your -- what does that mean, tolerance to respiratory depression?
A. So after you take a medication, and we'll specifically speak of opiates, within days to perhaps even as short as hours per se, you have an initial increase in your respiratory rate to the sedation and decreasing your breathing. So as a result of that, you will increase -- your body will make an adaptive response.

Over time, you tolerate the greater and greater levels of the carbon dioxide, that's tolerance to the respiratory effects. However, that is not a ceiling effect. Unlike with the pain medication effect that you're talking about, that you can continue to escalate the doses and perhaps never achieve pain management, but you are limited in the respiratory depressant effects of that drug. It will occur.
Q. So, on the one hand, there's no ceiling for prescribing opiates to treat pain, correct?
A. Correct.
Q. The FDA has not put a ceiling on how much you can give/prescribe, right?
A. Correct.
Q. All right. But, on the other hand, you're saying that a patient who has become tolerant on the medication also becomes

tolerant to respiratory depression?
A. Correct. But tolerance is not a ceiling effect for respiratory depression.
Q. Okay. What does that mean? Break it down.
A. The -- unlike the pain value, where you can continue to escalate, you can't continue to escalate towards eternity because at some point in time you will stop breathing because the carbon dioxide level continues to elevate and that oxygen level will decline.
Q. Okay. But as we speak, there's been no ceiling on prescribing dosages for opiates to treat pain, right?
A. The dosages have to be taken into account with the frequency of administration. The actual quantity dosages that you're speaking of, correct, there is no ceiling effect on -- to achieve that, based off of numbers. However, as I mentioned, the interval frequency of administering those drugs will be important with regards to that side effect profile.
Q. Okay. And we're going to get into that. I'm just trying to get to the very simple answer whether or not there's a ceiling of prescribing opiates by the FDA.
A. There is no quantity value associated with prescribing pain medicines to achieve pain relief. You are limited by the side effect profile.
Q. Okay. Now, in formulating your opinion that you have given today, one of the considerations has been the toxicology

report, correct?
A. It is one of a number of factors, correct.
Q. And you would agree with me that there are at least 58 pain relief drugs that contain oxycodone, correct?
A. You mean under multiple formulations that contain oxycodone?
Q. Yes, sir.
A. Yes.
Q. I'm saying it in a real --
A. I don't know the exact.
Q. -- elementary level so I can answer. But there are a number of drugs, pain relief medications, that contain oxycodone, right?
A. There are multiple formulations, correct.
Q. I mean, more than 50?
A. I don't know the exact number. There's -- if it has oxycodone in it, then it is oxycodone.
Q. So no matter what the level of oxycodone is in a medication, if it has oxycodone in it, it is oxycodone?
A. No, the milligram dosages would be different. I mean, the potency of that medication due to the milligram dosages would be different. But oxycodone is oxycodone.
Q. Okay. So I'm just trying to make sure that we're all on the same page as we continue.
There are various drugs that contain oxycodone, right?

A. Yes.
Q. And if it contains oxycodone, you would consider it to be oxycodone?
A. Yes.
Q. Okay. Now, there's no chemical test to distinguish this pill that may have oxycodone in it versus this pill that may have oxycodone in it, correct? When you see the tox result, it is just going to say oxycodone, right?
A. Yes.
Q. I would like to talk with you next about opiate blood levels.
A. Okay.
Q. Opioid blood levels. And when I say that term, what does that mean?
A. Are you speaking about premortem or postmortem?
Q. Because there's a difference, right?
A. Yes.
Q. What is antemortem?
A. Antemortem is before death.
Q. Okay. And what is postmortem?
A. After death.
Q. All right. Now, you would agree that prescribed opioids is an aggressive drug therapy, right?
A. According to the World Health Organization for pain management, yes, it is at a stepwise progression.

Q. And like any other area of medicine or therapy, there must be a method of measuring the effectiveness and proper usage of prescribed opiates, correct?
A. Yes.
Q. And determining blood levels is a primary way of doing that?
A. No.
Q. No? What's a primary way of doing that?
A. It is one of the ways. I would not necessarily say it is the primary way. If you have multiple drugs that you're taking simultaneously, a single concentration would not be indicative of toxicity.
Q. So determining blood levels is a way --
A. It is a way.
Q. -- of determining or measuring the effectiveness and the proper usage of opiate therapy?
A. I don't know about effectiveness nor the proper usage. It is a way of measuring.
Q. Okay. And the factor of the blood level has to be taken in consideration with the patient's condition, right?
A. As well as other drug therapy, correct.
Q. So you do look at the age and the sex of the person and the weight and the cause of pain and things like that, right?
A. Yes.
Q. You also look at how long they've been on the opioid drug

therapy?
A. Yes.
Q. And when you look at all of those factors, you can determine tolerance, can't you?
A. It is one of the potential mechanisms for determining tolerance, yes.
Q. And you would agree with me that looking solely at prescription amounts without blood level information to determine tolerance is inadequate, right?
A. No.
Q. So is it your testimony that you can determine tolerance by either just looking at blood levels or solely looking at prescription amounts?
A. No.
Q. You have to use both, right?
A. You assert that the blood level is the only mechanism by which to determine tolerance outside of the prescription. The blood level is the blood level. If there's other multiple medications simultaneously with it, that blood level value in isolation does not mean much.
Q. So I am not sure that you answered my question, which was: You would agree that looking solely at prescription amounts without the blood levels to determine tolerance is inadequate?
A. I can't answer your question the way you phrased it. If you're asking me, for example, with the oxycodone, if you have

an oxycodone blood level and you're on four other medications, does that single oxycodone value show tolerance? No. Does that value -- can it be interpreted, even if it is high or low, as toxic? No, because there's multiple mitigating factors with that.

Q. You also need to look at the person's entire medical file, right? To determine tolerance, that's what we're talking about.

A. It is significantly helpful, yes.

Q. The medical records?

A. Yes.

Q. Now, chronic pain patients can function and they may function well if they are tolerant to high dosages of opioids, right?

A. Are they only on opioids?

Q. Well, let's just -- in my question, yes.

A. Yes.

Q. And they can tolerate high dosages of opioids, we're talking about chronic pain patients, if they're on other medication, too, correct?

A. Yes.

Q. Why did you make me separate those out?

All right. Now, comparing chronic pain patients to, let's say, opioid naive patients, people who are not tolerant to high dosages, are you familiar with Forest Tennant's blood study on

opiate blood levels?
A. I read it.
Q. And that study deals with high dosages of opioids for chronic pain patients, right?
A. First -- yes, it does.
Q. And in that study, the opioid blood concentrations clearly showed that patients become tolerant to high opioid drug dosages and can physically function well?
A. I would not use the word "clearly."
Q. Okay. Let me take that word out and just say that that study showed that opioid blood concentrations show that patients who were tolerant -- and let me just rephrase that, because my question is becoming jumbled.
Tell me what that study showed.
A. First off, the study was conducted -- it was a horrible study.
Q. Why?
A. Well, several reasons: Number one, it is a nonindexed journal. It was not necessarily peer reviewed, per se. It was subjective reports by a few pain physicians who subjectively pulled data on a few patients.
There was no specific control for laboratory values, there was no specific single control on where they were sent. There was no characteristics and comparisons to other patients. They were subjective reports, asked for submission by the author of

that paper.
Q. And you've said that the author of the paper was a pain specialist?
A. I didn't say what he was. I didn't know. It is not an indexed journal. Until I received it, it was --
Q. Is it your testimony that Forest Tennant is not --
A. I don't know who he is.
Q. So I thought you just said that this was a paper and a study done by pain specialists.
A. Correct.
Q. Okay. And you're just saying you don't know who Forest Tennant is?
A. I'm saying that he was the author of the paper, if he was a pain physician, because he requested other pain physicians, yes. I don't know his training, background. It was not submitted in the paper.
Q. Okay.
A. But the paper asserts subjective conditions in subjective patients with isolated blood concentrations with a single drug.
Q. But it was a study done?
A. It was a study, yes. It was a subjective study written in a nonpeer reviewed journal that's not indexed in the National Library of Medicine. I could not pull that paper up at all.
Q. So obviously you didn't rely on it, correct?
A. Well, you can't compare the plasma concentrations of a

living person to a postmortem concentration.
Q. That's not my question.
A. I did not rely upon it, no.
Q. Now, you testified that various toxic levels or fatal blood concentration levels were indicated for various patients here, correct?
A. I reported the values that were obtained in the literature that I reviewed, and I reported what the concentrations were from the toxicology reports.
Q. I'm sorry. I'm trying to figure out, was that a yes?
A. Yes.
Q. And those levels, those toxic levels, you said, you know, in your report, and we're going to get to it, that the levels were toxic for certain individuals, right?
A. I stated that they were within the spectrum that was reported for toxic concentrations for that drug.
Q. And those levels, though, that were reported don't take into account dosage regimes for chronic pain patients, right?
A. Not all of them. The studies that I looked at did not necessarily differentiate between acute and chronic pain patients. The article by Spiller, et al., looked at findings specifically with regards to the hydrocodone and oxycodone concentrations. A subcategorization of those patients included therapeutic misadventure, which means they were prescribed this for a period of time. I don't know the chronicity of that. It

was not asserted in any of the papers, chronicity versus acute.
Q. So when you looked at the information that you researched to determine whether or not these levels were toxic or therapeutic, you can't say whether these levels involved or spoke to chronic pain patients, right?
A. I cannot exclude them nor include them completely. They were values reported for a range of patients that died as a result of suspected oxycodone or hydrocodone toxicity.
Q. So, Doctor Policastro, when you say you can't exclude them or include them, is that a fancy way of just saying I don't know?
A. Meaning exactly that, yes.
Q. So the comparative levels that you described in your testimony about what was toxic and what was therapeutic may actually be therapeutic levels for chronic pain patients or drug-tolerant patients, right?
A. Yes.
Q. Turning your attention to the toxicology reports.
A. Yes.
Q. You didn't examine any of the worksheets of the labs that wrote out the reports, correct --
A. No.
Q. -- that produced the results?
A. No, all I had was the report.
Q. So you basically accepted the report at face value, right,

because that's all you had?
A. Yes.
Q. Now, correct me if I'm wrong. Did I understand your testimony to be that while you looked at toxicology reports, they weren't significant in making your -- giving your opinion?
A. I didn't say they were significant or insignificant. I said they were one of the tools that I listed in evaluating the patients. There was a value reported, I reported what the value was. I showed when there was postmortem redistribution, I asserted that, and where was the sample if it was not a known sample site, which could also create artificial elevation.
Q. But it is true that you gave an opinion in some cases where you didn't even have a toxicology report, correct?
A. If there were factors --
Q. Is that a yes?
A. Yes.
Q. And, again, that's because your opinion is based on the qualitative synergistic effect of the drugs?
A. Yes.
Q. So the tox reports, since you didn't have them in some of the cases, really weren't your main focus, then, correct?
A. They were a component of them. They were not main or diminutive or excessive. They were there.
MS. CROSS: Your Honor, I think I'm good on time.

THE COURT: Okay.

Q. In the course of caring for living patients --

A. Yes.

Q. -- you often interpret hospital lab tests?

A. Yes.

Q. And in doing so, you can easily find out the normal value of a particular drug, right?

A. I'm not understanding your question.

Q. Well, when you interpret hospital lab reports, you can determine what the value of a particular drug is?

A. That doesn't mean it's toxic or not toxic.

Q. No, I'm just saying that you can determine what the -- you can have an accurate assessment of what the value is, that's all I'm asking.

A. They may report a range, they may not. But yes, most often time they report a range of normalcy.

Q. But when someone dies, the blood is not circulating, correct?

A. Correct.

Q. And so the noncirculating blood after death is not the same as the circulating blood when someone is alive, correct?

A. Correct.

Q. And a single blood concentration level in a living body can be different postmortem, correct?

A. Yes, yes.

Q. In other words, a single blood concentration level in a living person can be different than when they are dead?
A. Yes.
Q. And oftentimes those levels are different, correct?
A. Yes.
Q. In fact, studies have shown that postmortem blood, blood after you die, noncirculating blood, concentration levels don't correlate directly with blood concentrations before the person died?
A. Correct.
Q. Now, a lot goes into the interpretation of postmortem blood concentration levels, right?
A. Yes.
Q. And there's a phenomenon called postmortem redistribution?
A. Yes.
Q. And postmortem redistribution can actually distort to toxicology results, right?
A. Yes.
Q. And when we talk about postmortem redistribution, what we're talking about is drugs moving in the body when you're dead?
A. Yes.
Q. So drugs being in a certain place in the body when you're alive, but as soon as you decease, they move? That's what we're talking about, postmortem redistribution, right?

A. Yes.
Q. And you would agree with me that there is no proven mechanisms for determining postmortem redistribution?
A. Yes.
Q. It could be because of lividity or some other cause, right?
A. The factors that have been attributed to postmortem redistribution are dependent upon the characteristics of the drugs themselves is what has been assumed. I mean, yes, dependent positions and shifts in fluids could also create that and there's no specific single mechanism associated with it, correct.
Q. And so without adequate evaluation of, I guess, kinetic data, movable -- how drugs move, without any information about that, investigative information and consideration of postmortem changes, you could have misinterpretation, couldn't you?
A. Yes.
Q. It's important, you would agree, that -- it's important to interpret correctly those types of blood levels and postmortem redistribution, take that into consideration because it can just skew the results totally of toxicology reports, right?
A. Yes.
Q. Now, with chronic drug use or tolerance, tolerance cannot be measured or estimated after death, can it?
A. No.
Q. You can only determine tolerance when someone is alive,

right?
A. Yes.
Q. So if the blood concentration at the time of someone's death can't be known, then how is it possible to extrapolate the time and amount of drug ingested before death?
A. You cannot.
Q. You can't?
A. Correct.
Q. Can a medical examiner do that?
A. No one can do that. It is inaccurate to.
Q. Why?
A. Because the concentration plasma of an agent is equal to the amount of drug divided by the volume of distribution times the weight in kilograms. The volume of distribution drug postmortem is unknown. So therefore the plasma concentration before death cannot be accurately determined.
Q. Okay. And say that in layman's terms.
A. The characteristics of the drug that make the drug move around, that number is unknown at death. As a result of that, you cannot make an accurate determination of what that blood concentration would be before death.
Q. So this postmortem redistribution phenomenon is pretty important when you're trying to determine cause of death and you have drug blood levels, correct?
A. Yes.

Q. And in fact, after death, this phenomenon, postmortem redistribution, can elevate levels that are on basically the toxicology reports, right?
A. Yes.
Q. They can be skewed?
A. Yes.
Q. You would agree that most of the opioids prescribed by Doctor Volkman undergo postmortem redistribution, right?
A. Yes.
Q. Now, also in determining cause of death and, in conjunction, considering this postmortem redistribution, site sampling is important, too, right?
A. Yes.
Q. And when we say site sampling, we're talking about where the blood is drawn from out of the body, right?
A. Yes.
Q. And site sampling is very important too, because it can affect toxicology interpretation as well?
A. Yes.
Q. Just like postmortem redistribution?
A. Yes.
Q. And the closer you get to the heart with the site sample, the higher the blood level can be?
A. Correct.
Q. Right?

A. Yes.
Q. So without knowing the site sample, the place where the sample of blood was taken from on the body, without knowing that, you can't make a wholly accurate interpretation of the toxicology results, can you?
A. You can note the concentration and note the concerns for elevation, correct.
Q. So I understand you can note the concentration levels, but when you're interpreting the toxicology results, you can't be 100 percent accurate because you don't know where in the body the sample was taken from, right?
A. Yes.
Q. And in this case, the site sampling is unknown for James Estep, correct?
A. I would have to review my report. If you could provide me a copy, I could state that. But if I noted it in my report, if it was sample site unknown, I noted it.
Q. Okay. And if you noted it in the case of Steve Heineman, if it is in your report, then that's correct, right?
A. Yes.
Q. And if you noted it with regard to Charles Jordan, that the site sampling was unknown, if it is in your report, that would be correct?
A. Yes.
Q. Same thing with Dwight Parsons and Ernest Ratcliff, too,

right?
A. Yes.
Q. Now, you mentioned the vitreous fluid.
A. Yes.
Q. And you said that was the fluid behind the eye?
A. In simplistic terms, yes, that's why I said it that way, yes.
Q. Thank you, because I'm real simple.
A. I don't know about that.
Q. And so vitreous fluid concentration levels, well, you mentioned that on some of the deceased patients, right?
A. Correct.
Q. And the importance of testing it -- it is important to test vitreous fluid?
A. It provides another matrix for evaluation of a drug concentration.
Q. And it is important to test the vitreous fluid levels because that concentration level kind of lags behind the other sampling sites?
A. Yes.
Q. And it actually can give you a clearer picture in some instances because it does lag behind, the concentration level of vitreous fluid can, in some cases, give you a clearer picture?
A. It is a useful adjunct, so, yes.

Q. I mean, you do know that vitreous concentration levels are good or proven with alcohol studies, right?
A. Alcohol concentrations are 10 to 15 percent higher in the vitreous compared to whole blood samples because the percentage of water of the vitreous is 99 percent. So, the alcohol lags behind in the vitreous sample by approximately two hours, and it is 10 to 15 percent higher.
Q. So taking the concentration in the vitreous fluid for alcohol cases is a good idea or not?
A. It's present. As I just mentioned to you, it lags behind by approximately up to two hours. It is 10 to 15 percent higher because of the water weight. Whole blood has a specific factor difference compared to other fluid, plasma, vitreous, CFS. So to have a complete comparison analysis from the vitreous to blood is different.
Q. Okay. Now, there have been no studies that compare vitreous fluid concentrations to blood concentrations, right?
A. False.
Q. No? There are?
A. Yes.
Q. Tell me about them.
A. Well, there are several. It depends on the drugs that you're speaking of.
Q. We're talking about pain medication.
A. Okay. So in the Journal of Analytical Toxicology in 2009,

October, a comparison was made between oxycodone vitreous and oxycodone femoral, correlation factor was 1.17.

Q. Okay. 1.17, correlation factors. Is that what you said?

A. Vitreous to plasma, that value equals 1.17.

Q. What does that mean?

A. It means that they felt that they were to be equivalent -- when you have a numeric value of one and divide into two samples, it means that they're equivalent. There have been other studies that I can't recall off the exact top of my head, that have been performed and compared -- that evaluated vitreous oxycodone levels. That is the more recent study that I noted.

An article came out, and I don't recall the exact journal nor the date, on meprobamate concentrations that compared vitreous to femoral blood. So there are studies that compare vitreous to femoral, correct.

Q. And are they all fairly consistent with each other, the studies?

A. No. Because, again, the specific drug characteristics are different. Cocaine is higher in the vitreous compared to the blood.

Q. Before we go to the Power Point, I want to ask you, there are no interactions or contraindications for oxycodone 30, Xanax 2, and Soma, correct?

A. What do you mean there's no contraindications?

Q. Well, have you ever asked for a drug interaction result from a pharmacist? Or you don't need to do that because you're a toxicologist?
A. I would not specifically ask a pharmacist: Does Soma, Xanax and oxycodone interact? The answer to that is yes, they do.
Q. Okay. But do they -- is there a proven reference that they interact negatively?
A. Yes.
Q. And where do we get that, from your 34 references?
A. Yes.
Q. Okay.
A. The combination of opiates and sedatives produce an increase in risk for respiratory depression. This is a well recognized phenomenon.
Q. And so when you say respiratory depression, are you talking about immediate death?
A. It could or could not be. It depends on, as you mention, their level of tolerance, dosing frequency, intervals, individual genetic factors, multiple factors. But a combination of sedative and opiate dramatically increases their risk for adverse effect. When you look at patients that overdose, epidemiologically, those patients that died had most often combined effects.
Q. Now, when you speak about adverse effect, it appears that

you're only talking about death. But adverse effect could be something from a mild irritation or --
A. What do you mean, irritation?
Q. I'm trying to understand what you mean by adverse effect.
A. I mean an increase in likelihood for respiratory depression, sedation, mental clouding, one of those, in addition to other factors.
Q. So we've heard already that you would not prescribe oxycodone 30, Xanax and Soma together, right?
A. Correct.
Q. But you're not telling this jury that if anybody did that that it would automatically lead to respiratory depression?
A. No, I'm saying that the risk is dramatically increased versus those agents separate.
MS. CROSS: I would like to turn to the Power Point, Your Honor, if I may have assistance from the U.S. Attorney.
THE COURT: Okay.
Q. Doctor Policastro, is there any FDA contraindications for that combination of the drugs, Soma, oxycodone and Xanax?
A. I can't speak to what the FDA would list as a combination risk. They do not evaluate the combination of those agents.
Q. You don't know?
A. I do not know of any specific literature from the FDA regarding prescribers, do not prescribe those agents together.
Q. Yes, sir. Now, this Power Point that you testified as an

aid from, did you create it?
A. It was created with assistance of the U.S. Attorney. It was the information that I had.
Q. So what was your role in the Power Point?
A. What was my role in it?
Q. Yeah.
A. Talking about the information that was contained in it was a summation of my report.
Q. And then it was created for you?
A. Correct.
Q. Okay.
MS. CROSS: Now, can we turn to slide number seven.
Q. Doctor Policastro, you remember seeing this and testifying about this?
A. Yes.
Q. Now, you were talking in reference to this slide that if you dose the drug before the half-life, then you're putting what, more drug into the body before it can excrete it?
A. Yes.
Q. And is there some FDA rule or principle that you cannot prescribe that way?
A. It is pharmacologic principles.
Q. Okay.
A. I mean, it is the way you would dose medications. So there's no FDA rule per se, but it's pharmacologic principles

of the drugs.

MS. CROSS: May we have slide nine?

Q. You remember testifying about the effects of opiates on the bowels?

A. Yes.

Q. And you said that the bowels slow down as drugs come in?

A. No. I said the side effects of opiates cause bowel slowing.

Q. So it is a side effect?

A. Correct.

Q. And it is true, though, that the body has ways of compensating for this, though, right?

A. No.

Q. Not at all?

A. Not the constipation, no. There is no tolerance for constipation.

Q. Now, even though on this slide, still -- even though, I guess, material would go through the intestines slower, this doesn't change the total absorption of the drug, though, correct?

A. No.

Q. In fact, oxyContin is designed to be slowly released as it passes through the gastrointestinal system or bowel system?

A. Yes.

MS. CROSS: Slide 10.

Q. Now, if you wouldn't mind, explain what you meant by the bullet point that says, But cannot do anything about it. Can you just explain that, please?
A. Yes. So even patients that have tolerance continue to have elevations in their carbon dioxide level.
Q. The air that comes out?
A. Correct. As you accelerate dosing of narcotics, the capacity to respond to that change in carbon dioxide increases. The body can only make so much change in response to its carbon dioxide level.
Q. Okay. So you're talking about what the body can do, the effect that the body -- it has on the body, right?
A. Correct.
Q. But you do agree that in situations like that, more oxygen can be given to the patients, right? The patient could be put on oxygen?
A. That doesn't change the carbon dioxide level or the other risks for respiratory depression. Oxygenation and ventilation are separate. They are also interdependent, but they're separate. The problem is that as patients' carbon dioxide level increases and as they have continued respiratory decline, that oxygenation will drop. Their capacity to respond to a triggered event, if that oxygen level goes down, to kickstart the breathing, is gone as the opiate dosing increases. So just because you oxygenate someone does not get rid of their carbon

dioxide level.
Q. And have you heard of Narcan?
A. Of course.
Q. What is that?
A. It is an opioid antagonist.
Q. So basically when that is administered --
A. It reverses the effects.
Q. -- it reverses the effect of the opiates, right?
A. Correct.
Q. So when we look at this and when you were testifying, you were testifying about what the body cannot do, not what other methods can be employed by medical personnel --
A. Correct, correct.
Q. -- to reverse this?
A. Correct.
Q. Okay. Because there are other things that can be done to reverse respiratory depression?
A. To a point, correct.
MS. CROSS: If we could look at slide 17.
Q. This is in regard to, I believe it is, Dwight Parson --
A. Yes.
Q. -- you testified about. And you were looking at the -- you called it a toxicological chaos?
A. I did.
Q. And that was because of the dosage regime?

A. Yes.
Q. Now, this is a prescription -- or not a prescription, but a progress note for what date?
A. I would have to review my records. If you provide them to me, I can --
Q. If you can just look on there, do you see the date on the top of one of the pages?
A. 5/12/04, I'm sorry.
Q. So that's a progress note for May 12th, '04, right?
A. Yes.
Q. If Dwight Parsons had been on this dosage regime since April of '03, then why is it a problem now?
A. He still died.
Q. So if he was on the same regime a year earlier, you would be saying he's going to die?
A. I'm saying did he take all of those medications in that exact order? Was he compliant with that schedule?
Q. So what you need to know is if he actually took those medicines?
A. Correct.
Q. And you don't know?
A. Correct.
Q. But yet, you said that the medicines killed him?
A. I'm stating that that schedule of medicines that was prescribed like that, with the combination of those agents,

could produce adverse effects, yes.
Q. So it is important in each of the cases that you have opined about death to know whether or not the person even took the medication, right?
A. Correct.
Q. And you don't know, do you?
A. Well, if there was a toxicology analysis that showed that they had the drug present, then they took the drug.
Q. You don't know how many pills, though, right?
A. Correct.
MS. CROSS: Looking at slide 19.
Q. This is in regard to red flags you said for Paul Crum?
A. Yes.
Q. And you noted that he had several medical issues that made him vulnerable to respiratory depression?
A. Yes.
Q. So looking at someone like this, who is vulnerable, if they are an established chronic pain patient, do you not treat their pain?
A. I never suggested not to treat their pain. What I suggested is that the combination of the agents dramatically increases his risk for further respiratory decline.
MS. CROSS: Turning to slide 20.
Q. This was a prescription you testified given by Doctor Volkman in December of '05, right?

A. Yes.
Q. And you looked at Mr. Crum's medical chart, didn't you?
A. I did.
Q. And you know that he was on the same regimen, dosage regimen, for several months before this one, correct?
A. Correct. What I don't recall, I would have to look at the records, was the amount and dosing per day, if that had changed.
Q. If it were the same for several months prior, it is your testimony that this is the prescription, if he took the medication, because we don't know, right? We don't know what he took?
A. Correct.
Q. That this is what killed him?
A. I'm stating that when you accelerate the dosages and the combination of those agents, that that would produce respiratory depression.
Q. What if the regime was the same, the same dosages, the same amount, the same type of medication months earlier? Would that still be your opinion?
A. I'm saying that he has severe oxygen dependency, COPD, he has a significant lung disease, those would be a contributing factor towards his risk for respiratory depression.
Q. Turning to slide 30, Aaron Gillespie. You were not given any investigative reports to look at, were you, Doctor

Policastro?
A. Whatever was noted, then I noted. I don't recall any other additional investigative reports.
Q. Okay. And so you looked at the prescription history for Aaron Gillespie, right?
A. Yes.
Q. And you noted the number of pills that he was prescribed on that day, correct?
A. May I see a copy of my report?
Q. Do you have one in the courtroom?
A. I was told that I had to ask the clerk to have one.
THE COURT: Mr. Wright apparently has one.
MS. CROSS: May I?
THE COURT: You may. I just note that it is 4:30 and maybe it would be a wiser use of our time to give the doctor an opportunity to review over the evening hours.
Ladies and gentlemen, let's break for the day. And I remind you not to discuss the case among yourselves or with anyone else or permit anyone to discuss it with you or in your presence. Report any violation to Ms. Brown. Make no attempt to do any research or investigation on the case, persons involved in the case or any of the issues in the case over the evening hours. And no Internet chitchat of any kind regarding the case.
We'll see you back at 9:00 tomorrow morning.

THE COURTROOM DEPUTY: All rise.

(The jury left the courtroom at 4:30 p.m.)

(The following transpired out of the presence of the jury.)

THE COURT: Doctor Policastro, you're excused for the evening.

THE WITNESS: Okay.

THE COURT: See you back at 9:00 tomorrow morning.

THE WITNESS: Yes, ma'am.

THE COURT: No discussion of your testimony with anyone over the evening hours.

THE WITNESS: Yes, ma'am.

THE COURT: Counselors, anything you would like to put on the record in the absence of the jury before we take the break?

MR. WRIGHT: No, Your Honor.

MS. CROSS: No, Your Honor.

THE COURT: All right. See you back here at 8:30 tomorrow.

THE COURTROOM DEPUTY: All rise.

(Proceedings concluded at 4:34 p.m.)

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I N D E X

C E R T I F I C A T E

I, Jodie D. Perkins, RMR, CRR, the undersigned, certify that the foregoing is a correct transcript from the record of proceedings in the above-entitled matter.

s/Jodie D. Perkins
Jodie D. Perkins, RMR, CRR
Official Court Reporter